IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY

TAKEDA PHARMACEUTICALS AMERCA, INC., et al., **Plaintiffs,** **v.** **APOTEX, INC.,** **Defendant.**	Civ. No. 21-12998 (KM) (AME) **OPINION**

KEVIN MCNULTY, U.S.D.J.:

This patent infringement case is brought by Takeda Pharmaceuticals America, Inc., Takeda Pharmaceuticals U.S.A., Inc., and Ariad Pharmaceuticals, Inc. (collectively, "Takeda") against Apotex Inc. ("Apotex"). The patents-in-suit are Patent Nos. 9,493,470 (the "'470 patent"), 11,192,895 (the "'895 patent"), 11,192,897 (the "'897 patent"), and 11,384,086 (the "'086 patent").[1] These patents are directed to crystalline forms of ponatinib hydrochloride and their use to treat certain types of leukemia.

Takeda commenced this infringement action after Apotex sought approval for a generic ponatinib hydrochloride tablet. This Opinion contains the Court's construction of a key patent term following a *Markman* hearing.[2]

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[1] On November 3, 2022, this action—which previously involved only the '470, '895, and '897 patents—was consolidated with Civil Action No. 1:22-cv-06151, a parallel patent infringement case between the same parties involving the related '086 patent. (DE 103.) Pursuant to the parties' November 21, 2022 stipulation (DE 107), the resolution of this claim construction proceeding regarding the '470, '895, and '897 patents, which was already underway at the time of consolidation, will apply as well to the claims of the '086 patent.

[2] The reference is to *Markman v. Westview Instruments, Inc.*, 517 U.S. 370 (1996).

I. Background

The invention claimed by the patents-in-suit consists of crystalline forms of ponatinib hydrochloride and associated treatment methods. Ponatinib hydrochloride, the active ingredient in Takeda's Iclusig product, is a cancer drug used to treat two types of leukemia: 1) chronic myeloid leukemia, also known as chronic myelogenous leukemia ("CML"), and 2) Philadelphia chromosome-positive ("Ph+") acute lymphoblastic leukemia ("ALL," and together, "Ph+ALL"). (Apotex PPT at 28.)[3] CML originates in granulocytes, a type of white blood cell, and is divided into three phases of progression: chronic phase, followed by accelerated phase, then blast phase. (*Id.*) Ph+ALL, on the other hand, originates in lymphocytes, another type of white blood cell. (*Id.*)

"Philadelphia chromosome" refers to an abnormally small chromosome 22 that may present in patients with CML and ALL. (*Id.* at 29.) This genetic mutation, first discovered in Philadelphia, is the result of a translocation between chromosome 9 and chromosome 22 in humans. (*Id.* at 29-30.) This translocation yields a Bcr-Abl fusion gene, which codes for the Bcr-Abl fusion protein. (*Id.* at 31.) Normal Abl protein, a tyrosine kinase enzyme, regulates cell division and survival. (*Id.*) However, when Abl protein is fused to Bcr protein, Abl acts abnormally, leading to increased cell division and survival of certain blood cells, which replace normal blood-forming cells. (*Id.*)

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[3] Certain key items from the record will be abbreviated as follows:
DE = Docket entry number in this case
Compl. = Takeda's First Amended Complaint (DE 34)
Takeda Br. = Takeda's opening claim construction brief (DE 63)
'470 Patent = Patent No. 9,493,470 (DE 63-2)
'895 Patent = Patent No. 11,192,895 (DE 63-3)
Apotex Br. = Apotex's opening claim construction brief (DE 65)
Takeda Resp. Br. = Takeda's responsive claim construction brief (DE 78)
Apotex Resp. Br. = Apotex's responsive claim construction brief (DE 79)
Takeda PPT = Takeda's PowerPoint presentation from *Markman* hearing
Apotex PPT = Apotex's PowerPoint presentation from *Markman* hearing

Treating CML and Ph+ALL centers around inhibiting the Bcr-Abl fusion protein. (*Id.* at 35.) Researchers have developed drugs known as tyrosine kinase inhibitors ("TKIs") that bind to and inhibit the Bcr-Abl fusion protein. (*Id.* at 36.) The first Bcr-Abl TKI that was developed for this purpose is imatinib, and it is considered a "first line" treatment. (*Id.*) However, a slight mutation in the Bcr-Abl fusion gene—namely the M351T mutation—can prevent imatinib from binding to the protein and therefore prevent it from inhibiting Bcr-Abl activity. (*Id.*) "Second line" TKIs, including dasatinib and nilotinib, were subsequently developed to treat imatinib-resistant patients. (*Id.* at 37.) Another genetic mutation in the Bcr-Abl fusion gene, however—the T315I mutation—may cause resistance to these second line TKIs as well, a problem the invention claimed by the patents-in-suit is intended to solve. (*Id.*) Ponatinib is a "third line" TKI that was developed to treat patients with this additional type of mutation. (*Id.*) Takeda's Iclusig, an embodiment of the patents-in-suit, is a ponatinib hydrochloride formulation administered in tablet form to CML and Ph+ALL patients who show resistance to first and second line TKI therapies. (Takeda PPT 15.)

According to the First Amended Complaint, filed on December 17, 2021, Apotex submitted to the FDA an Abbreviated New Drug Application ("ANDA") No. 215893, seeking approval to engage in the commercial manufacture and sale of generic ponatinib hydrochloride tablets. (Compl. ¶1.) Takeda alleges that the Apotex product infringes the patents-in-suit and seeks appropriate relief.

The Court held a *Markman* hearing on November 30, 2022. (DE 108.) Prior to the hearing, the parties submitted opening briefs, as well as briefs in response. (DE 63, 65, 78, 79.) I am now prepared to rule on the meaning of the disputed claim term.

II. Legal Standards

A patent infringement case involves two steps. First, the court determines the meaning of the claims in the patent. *Amgen Inc. v. Amneal*

Pharms. LLC, 945 F.3d 1368, 1375 (Fed. Cir. 2020). Second, the court compares the claims, as construed, to the allegedly infringing product. *Id.*

We are now concerned with the first step, known as claim construction. Where, as here, the parties dispute the meaning of the patent's claims, resolution of those disputes is an issue for the court. *Bayer Healthcare LLC v. Baxalta Inc.*, 989 F.3d 964, 977 (Fed. Cir. 2021). This task primarily requires construal of written documents (quintessentially, the patent itself), but some factual determinations may be needed to assist in understanding the written words. *Teva Pharms. USA, Inc. v. Sandoz, Inc.*, 574 U.S. 318, 325–26 (2015). Accordingly, there is a hierarchy of sources to be considered when construing a claim, arranged in decreasing order of importance. *Profectus Tech. LLC v. Huawei Techs. Co.*, 823 F.3d 1375, 1380-81 (Fed. Cir. 2016).

Of course, I "begin with the words of the claims themselves." *Allergan Sales, LLC v. Sandoz, Inc.*, 935 F.3d 1370, 1373 (Fed. Cir. 2019) (citation omitted). Those words receive the meaning that "a person of ordinary skill in the art" ("POSA") would give them. *Id.* (citation omitted). A POSA would interpret the words in the context of the rest of the patent document, including the specification which describes the invention. *Id.* at 1373 & n.6. The prosecution history, *i.e.*, proceedings before the U.S. Patent and Trademark Office that led to approval of the patent, can further illuminate the meaning of a term. *Id.* at 1373 & n.7. All of the foregoing constitutes "intrinsic evidence," *i.e.*, evidence from within the patent process itself.

I may also turn to "extrinsic evidence," or evidence outside the patent and prosecution history. *Id.* at 1373 & n.8. Such extrinsic evidence includes "expert and inventor testimony, dictionaries, and learned treatises." *Phillips v. AWH Corp.*, 415 F.3d 1303, 1317 (Fed. Cir. 2005) (en banc). In general, however, extrinsic evidence is less reliable than the patent and prosecution history. *Id.* at 1318. For that reason, extrinsic evidence is second-priority, and cannot "trump the persuasive intrinsic evidence." *Immunex Corp. v. Sanofi-Aventis U.S. LLC*, 977 F.3d 1212, 1221–22 (Fed. Cir. 2020) (citation omitted).

III. Discussion

The parties have identified a single claim term that requires construction by the Court: "subject." Takeda argues that the Court should construe "subject" to mean "human," whereas Apotex argues that the Court should construe "subject" to mean "human or other animal." (Takeda Br. at 1; Apotex Br. at 1.) Each party contends that its proposed construction is supported by 1) the claim language, 2) the specification, 3) the prosecution history, and 4) extrinsic evidence. I analyze each of these sources in turn.

a. Claim Language

Takeda's proposed construction is more directly aligned with the claim language. While Apotex is correct that, standing alone, the plain meaning of "subject" is not definitionally limited to "human" (Apotex PPT at 50), I must consider "the context of the surrounding words of the claim" in order to "determin[e] the ordinary and customary meaning" of the term as it appears in the patent.[4] *Brookhill-Wilk 1, LLC v. Intuitive Surgical, Inc.*, 334 F.3d 1294, 1299. Two elements of the surrounding claim language strongly suggest that the term "subject" refers specifically to a human subject:

First, the claims recite methods of using ponatinib hydrochloride to treat a condition that occurs only in humans. Specifically, claims of the patents-in-suit recite "method[s] for treating [Ph+ALL] in a *subject* in need thereof . . . " (*See, e.g.*, '470 Patent at claims 8, 9, 11, 16, and 17 (emphasis added).) As I explained *supra*, "Ph+" refers to the presence of the Philadelphia chromosome, a shorter-than-normal human chromosome 22 that results from translocation of the Abl gene from human chromosome 9. (Apotex PPT at 28.) Apotex concedes that the "Philadelphia chromosome" is a mutated chromosome 22

[4] I note that the parties do not agree on the POSA definition as it relates to this claim construction proceeding. Consistent with the parties' dispute over whether "subject" as it is used in the patents-in-suit covers nonhuman animals, Takeda takes issue with Apotex's inclusion of a veterinary doctor in its POSA definition. (Takeda Resp. Br. at 2-4.) However, Apotex has conceded that the claim construction dispute does not turn on the POSA definition. (Apotex PPT at 46.) I agree, and I decline to address the POSA definition dispute further.

originally discovered in humans but argues that the term refers "more generally to a human or other animal chromosome containing the abnormal Bcr-Abl fusion gene." (Apotex Resp. Br. at 4.) In other words, Apotex's position is that the term "Ph+" and "Bcr-Abl positive" are interchangeable. (Apotex Resp. Br. at 8.) In support, Apotex points to evidence that the presence of the Bcr-Abl fusion gene is the "hallmark" of diagnosing Ph+ALL, and that this gene may result from an equivalent chromosomal translocation in nonhuman animals. (*Id.* at 6.) I do not find these arguments compelling. The mere fact that identifying the Bcr-Abl fusion gene—which can occur in other animals—is a necessary condition to diagnosing the Philadelphia chromosome, *i.e.* a mutated human chromosome 22, does not necessarily mean that a POSA would understand the term "Philadelphia chromosome" to cover a nonhuman chromosome containing the Bcr-Abl fusion gene. Indeed, none of the literature on which Apotex relies refers to a nonhuman animal with a Bcr-Abl fusion gene as possessing a Philadelphia chromosome. In fact, according to literature discussed by both parties, when the Bcr-Abl fusion gene was first identified in dogs, the North Carolina State University researchers who made the discovery gave the canine equivalent of the Philadelphia chromosome its own geographical name; predictably, they named it the "Raleigh chromosome."[5] (Takeda Br. at 8; Apotex Resp. Br. at 6.) Prevailing research at the time of prosecution, then, used nomenclature *other than* "Philadelphia chromosome" to signify a nonhuman chromosome containing the Bcr-Abl fusion gene. This accepted usage further suggests that the terms "Ph+" and "Bcr-Abl positive" are *not* interchangeable and that the claim language regarding treatment of subjects that are Ph+ refers exclusively to treatment of humans.

[5] Unlike the Philadelphia chromosome, which is a shortened human chromosome 22, the Raleigh chromosome is a mutant dog chromosome 26. (Takeda Br. at 8; Apotex Resp. Br. at 6.)

Second, the claims recite "method[s] for treating" Ph+ALL and various phases of CML in a subject that is resistant or intolerant to prior TKI therapy.[6] According to the scientific evidence presented, this could only describe treatment administered to a human. Whereas human-specific first and second line TKI therapies are cited in the patent (*see* Section III.b., *infra*), Apotex has offered no evidence of animals receiving *treatment* in the form of TKI therapy. Rather, the literature on which Apotex relies involves administration of ponatinib hydrochloride to nonhuman animals only for purposes of pharmacology testing and research. For instance, Apotex cites intrinsic references to the O'Hare[7] and Huang[8] papers and argues that they "disclose mice that mimic CML resistance to TKIs by way of a mutated form of the Bcr-Abl protein normally responsible for CML." (Apotex Resp. Br. 10-11.) But these references do not suggest that TKIs are used to *treat* diseases in mice, whether successfully or unsuccessfully. Rather, they involve the implantation of cancerous cells expressing human Bcr-Abl into laboratory mice for experimentation. (Takeda Resp. Br. at 15.) Apotex also relies on the Wiley[9] reference, which, according to Apotex, "taught that imatinib could be used as a

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6 *See, e.g.,* '470 Patent claims 8, 9, 11, 16, 17 (identifying the invention as "[a] method of treating Philadelphia chromosome positive acute lymphoblastic leukemia in a subject in need thereof comprising administering to the subject . . . ponatinib . . . " as well as "[a] method of treating chronic myeloid leukemia in a subject in need thereof comprising administering to the subject . . . ponatinib . . . "); *see also* '895 Patent claims 2, 3, 8, 9, 14, 15, 20, and 21 (identifying the invention as a treatment for Ph+ALL or CML "in a subject in need thereof . . . wherein the subject is resistant or intolerant to at least one prior tyrosine kinase inhibitor" or "wherein the subject is resistant or intolerant to at least two prior tyrosine kinase inhibitors").

7 O'Hare et al., "AP24534, a Pan-BRC-ABL Inhibitor for Chronic Myeloid Leukemia, . . . Mutation-Based Resistance'. 16 Cancer Cell 401-12 (2009). (DE 64-6.)

8 Huang et al., "Discovery of 3-[2-(Imidazo [1,2-b]pyridazin-3-yl)ethynl]-4-methyl -N-{4-[(4-methylpiperazin-1-y1)-methyl]-3-(triftuoromethyl)phenyllbenzamide (AP24534), a Potent, Orally Active Pan-Inhibitor of Breakpoint Cluster Region-Abelson (BCRABL) Kinase Including the T315I Gatekeeper Mutant," 53 Journal of Medicinal Chemistry, 4701-19 (2010).

9 Jennifer L. Wiley, et al., "Chronic Myelogenous Leukemia in a Great Horned Owl (Bubo virginianus), 23(1) J Avian Med. & Surgery 36 (2009).

first-line therapy in owls just as it is in humans." (Apotex Resp. Br. at 11.) However, this teaching by the "Wiley" authors was conjecture; the great horned owl they examined, and presumed to have suffered from CML, died before they could put their hypothesis to the test and administer any TKI therapy. (Takeda PPT at 30.) Failing to demonstrate anything more than the speculative or "potential administration" of TKIs for veterinary treatment (Apotex Resp. Br. at 11), these sources do not support the proposition that the claim language regarding treatment of a subject that is resistant or intolerant to prior TKI therapy can refer to nonhuman animals.[10]

In sum, Apotex's proposed construction is irreconcilable with claim language that specifically recites treatment methods of subjects who are 1) Philadelphia chromosome positive, and 2) have previously undergone one or more rounds of TKI therapy, as these qualities could only describe human subjects.

b. Specification

The patent specification lends support to Takeda's proposed construction as well. Consistent with my finding *supra* that a "subject" who underwent prior TKI therapy must refer to a human subject, the specification's description of such prior therapies is likewise limited to specific human treatments. The specification provides:

> Ponatinib hydrochloride is a small molecule pan-BCR-ABL inhibitor in clinical development for the treatment of adult patients

[10] Takeda also argues that the claim language supports its proposed construction because classification of CML into chronic, accelerated, and blast phases only occurs in humans. (Takeda Br. at 8.) In support, Takeda cites criteria set forth by the World Health Organization ("WHO") for diagnosing accelerated and blast phases of CML in humans. (*Id.*) Setting aside the fact that the WHO's promulgation of guidelines for diagnosing a human condition does not mean that that same condition cannot present in nonhuman animals, Apotex cites literature that specifically used the same WHO criteria to diagnose phases of CML in dogs. (Apotex Resp. Br. at 9-10.) In any event, while I am unconvinced by this alternative argument, the claim language, particularly as it relates to the patents-in-suit directing treatment of subjects who are Ph+ and show resistance to prior TKI therapy, is still consistent with Takeda's proposed construction.

> with chronic phase, accelerated phase, or blast phase CML or Philadelphia chromosome positive acute lymphoblastic leukemia (Ph+ ALL) resistant or intolerant to prior tyrosine-kinase inhibitor therapy. Other tyrosine-kinase inhibitors relevant to such CML or Ph+ALL therapy include GLEEVEC® (imatinib mesylate) and TASIGNA® (nilotinib) (both from Novartis AG), SPRYCEL® (dasatinib) (from Bristol Myers Squibb Company) and BOSULIF® (from Pfizer Inc). A New Drug Application (NDA) for ponatinib hydrochloride was filed with the United States FDA on Jul. 30, 2012.

(470 Patent at 2:49-59.) Adopting Apotex's proposed construction of subject to mean "human or other animal" would lead to an illogical conclusion: *i.e.,* that ponatinib hydrochloride is properly used to treat nonhuman animals who have been previously administered one or more of the recited drugs, many or all of which are approved specifically for human use.

Apotex argues, however, that the patent drafters themselves used "subject" interchangeably with "humans or other animals," citing the following paragraph:

> [V]arious solid forms of ponatinib hydrochloride, may be administered . . . at a therapeutically effective amount to a subject in need of treatment. Similarly, any of the solid forms of . . . ponatinib hydrochloride disclosed herein may be formulated . . . into a pharmaceutical composition that can be subsequently used to treat various desease [sic] states in *humans or other animals*. For example, pharmaceutical compositions comprising any single one or combination of polymorphs of ponatinib and/or ponatinib hydrochloride may be used for treating CML or Ph+ALL in a subject in need thereof, by the administration of a therapeutically effective amount of the pharmaceutical composition to the subject in need thereof.

(Apotex Br. at 8 (quoting '470 Patent at 2:49-59) (emphasis added).) I do not agree with Apotex's reading of the cited excerpt. The reference to "other animals" (the only such reference in the patent), does not describe "other animals" suffering from Ph+ALL or CML, or "other animals" who proved resistant to first and second line TKI therapies—*i.e.,* the conditions the invention was created to treat. The reference to "other animals" appears in the

broader statement that ponatinib hydrochloride may be used more generally to develop pharmaceutical products for treating human or animal diseases. The following sentence, beginning "[f]or example," cites the treatment of a subject with CML or Ph+ALL as a *specific claim* of pharmaceutical use. A plain reading does not suggest that here, the "subject" must incorporate "human or other animal" from the preceding, more general sentence. My reading is corroborated by the specification's description of the method-of-treatment aspects of the invention, all of which specifically entail the treatment of a human subject:

> In another aspect, the present disclosure is directed to a method of treating a disorder or condition in a *human* that responds to the inhibition of a protein kinase by administering to the human a therapeutically effective amount of a polymorph of ponatinib disclosed herein. In certain embodiments, the disorder or condition is chronic myeloid leukemia (CML).
>
> In another aspect, the present disclosure is directed to a method of treating a disorder or condition in a *human* that responds to the inhibition of a protein kinase by administering to the human a therapeutically effective amount of a substantially pure crystalline form of ponatinib hydrochloride disclosed herein. In certain embodiments, the disorder or condition is chronic myeloid leukemia (CML) or Philadelphia chromosome-positive acute lymphoblastic leukemia (Ph+ALL) when the protein kinase is Bcr-Abl or a mutant form thereof.

('470 Patent at 3:50-4:31) (emphasis added).) The language of the specification therefore weighs in favor of Takeda's proposed construction.[11]

c. Prosecution History

The prosecution history of the patents-in-suit provides additional intrinsic evidence that supports Takeda's proposed construction. Specifically, it

[11] Apotex argues that the phrase "[i]n another aspect" is "non-limiting language" implying that the excerpted portions of the specification regarding humans do not foreclose the possibility of other aspects of the invention involving treatment of nonhuman animals. (Apotex Resp. Br. at 13-14.) This argument is unconvincing if only because the specification makes no mention of any aspect that specifically pertains to nonhuman animals.

contains a reference to the National Cancer Institute's Dictionary of Cancer Terms that defines the "BCR-ABL fusion gene" in human-specific terms:

> A gene formed when pieces of chromosomes 9 and 22 break off and trade places. The ABL gene from chromosome 9 joins to the BCR gene on chromosome 22, to form the BCR-ABL fusion gene. The changed chromosome 22 with the fusion gene on it is called the Philadelphia chromosome. The BCR-ABL fusion gene is found in most patients with [CML], and in some patients with [ALL] or acute myelogenous leukemia (ALL).

(Takeda Br. at 13 (quoting '470 Patent file history.) Recall that Apotex's position is that Ph+ can refer to any animal with a Bcr-Abl fusion gene. (Apotex Resp. Br. at 8.) The intrinsic record, however, defines the Bcr-Abl fusion gene and the Philadelphia chromosome as human chromosomal abnormalities. Apotex responds by arguing that the National Cancer Institute's Dictionary of Cancer Terms "is a lay, non-technical dictionary that would not have been relied on by a [POSA] to determine the meaning of a highly technical term such as this one." (Apotex Resp. Br. at 15.) I do not suggest that this dictionary definition is dispositive, but it is supportive of Takeda's position. An intrinsic reference to a dictionary definition can give meaning to related claim terms and provide useful context for the invention claimed by the patents-in-suit. Apotex contends that "this dictionary is intended to be used by the average person to achieve a basic understanding of cancers afflicting humans" (*id.*), but the supposed "intended" use of the dictionary is of no consequence. The dictionary was used by the inventors to communicate the scope of the patents-in-suit to the United States Patent and Trademark Office. Because this dictionary definition, referenced in the file history, limits that scope to treatment of a human-specific condition, it supports Takeda's proposed construction.

Apotex, for its part, cites other portions of the prosecution history to support its proposed construction. Apotex notes that the cited Huang paper (*see supra*, n. 9), as well as Zou,[12] define "subject" to include nonhuman

[12] Zou et al., U.S. Patent No. 8,114,874 (DE 64-5).

species. It follows, says Apotex, that "subject" as it appears in the patents-in-suit must mean "human or other animal." (Apotex Resp. Br. at 8-9.) This argument ignores the context of "subject" as it used in those references. For instance, the term "subject" in Huang is defined as a "human or another mammal . . . that can be afflicted with or is susceptible to a disease or disorder"—a context far broader than that of the "subject" in the patents-in-suit, who has undergone prior TKI therapy to treat Ph+ALL or CML. (Takeda Resp. Br. at 13.) Apotex also contends that Huang, Zou, and O'Hare (*see supra*, n. 8) "disclose the administration of ponatinib to treat cancers in animals including, but not, limited to, humans." (Apotex Resp. Br. at 9-10.) But as Takeda aptly points out, 1) the disclosures in Zou "are not specific to ponatinib, but rather apply to one or more of the thousands of compounds disclosed therein," and 2) Huang and O'Hare disclose the study of laboratory mice implanted with cells expressing human Bcr-Abl in order to study treatment for CML in humans, not the administration of ponatinib for veterinary treatment. (Takeda Resp. Br. 14-15.)

The prosecution history of the patents-in-suit, then, while not wholly unambiguous, generally supports Takeda's proposed construction.

d. Extrinsic Evidence

Because the intrinsic record strongly supports Takeda's proposed construction, I need not give great weight to the extrinsic evidence presented by either party, which cannot "trump the persuasive intrinsic evidence." *Immunex Corp.*, 977 F.3d at 1221–22 (citation omitted). Nevertheless, I will briefly take up the extrinsic evidence proffered by Apotex, which I find unpersuasive.

First, Apotex points to "technical and general purpose dictionaries at the effective filing dates of the patents-in-suit" that define "subject" as a "person or animal subjected to treatment, observation, or experiment" and a "person or animal that is the object or medical or scientific study." (Apotex Br. at 11.) Takeda does not dispute that the word "subject," as a matter of plain English, *could* include nonhuman animals in certain contexts; rather, Takeda's position

is that the term excludes nonhuman animals within the context of the patents-in-suit. Extrinsic dictionary definitions are not useful for the purpose of claim construction where, as here, they contradict a more specific, context-specific definition "ascertained by a reading of the patent documents." *Kaneka Corp. v. Xiamen Kingdomway Grp. Co.*, 790 F.3d 1298, 1304 (Fed. Cir. 2015) (quoting *Phillips v. AWH Corp.*, 415 F.3d 1303, 1322-23 (Fed. Cir. 2005)).

Second, Apotex cites a litany of prior art references which, it says, "taught that cancer falling within the scope of the claims can occur in animals in addition to humans." (Apotex Br. at 11.) Takeda spills considerable ink disputing the merits of this claim as it pertains to Apotex's many references (Takeda Resp. Br. at 16-19), but these publications fail to support Apotex's proposed construction for two simple reasons: None of the references describe a nonhuman animal as 1) presenting with a Philadelphia chromosome, or 2) having undergone one or more prior rounds of TKI therapy (*see* Section III.a., *supra*).

Third, Apotex points to Takeda's usage of the term "subject" in the course of obtaining other, unrelated patents as evidence that the term is not limited to humans when used in the patents-in-suit. Specifically, Apotex argues by negative implication that because Takeda elsewhere used specific limiting phrases such as "human subject" and "mammalian subject," it could not have intended such a limitation here. (Apotex Br. at 13.) Apotex's reasoning is both legally and factually flawed. As a threshold matter, statements made in unrelated patent applications are not relevant to claim construction.[13] *See Apple Inc. v. Motorola, Inc.*, 757 F.3d 1286, 1312 (Fed.Cir.2014), reversed in part on other grounds, *William v. Citrix Online, LLC*, 792 F.3d 1339

[13] It is for this same reason that Apotex's reliance on *Vifor (Int'l) AG v. Mylan Labs. Ltd.*, No. 19-13955, 2021 WL 2652123 (D.N.J. June 28, 2021) is misplaced. Apotex essentially argues that because, in another case involving an unrelated patent, "this District construed the word 'subject' to not be limited to humans," I should follow suit with respect to the term's usage here. (Apotex Br. at 10.) It would be patently improper for me to do so.

(Fed.Cir.2015) (en banc). Even if it were proper to consider such statements, however, the unrelated patents Apotex analyzes were assigned to different corporate entities at the time they were prosecuted. Apotex's suggestion that the Court construe "subject" as it is used in the patents-in-suit consistent with its usage in unrelated patents assigned to a distinct corporate entity is therefore premised on a faulty comparison.

The extrinsic evidence presented by Apotex does little to undermine Takeda's proposed construction, which is clearly supported by the intrinsic record.

* * *

Because the weight of the intrinsic evidence supports Takeda's position that the patents-in-suit are directed to human-specific treatment, I adopt Takeda's proposed construction of "subject" as "human."

IV. Conclusion

I construe the disputed term "subject" as follows: "human."

A separate order will issue.

Dated: January 13, 2022

/s/ Kevin McNulty

Hon. Kevin McNulty
United States District Judge